Periprocedural Predictors of New-Onset Conduction Abnormalities After Transcatheter Aortic Valve Replacement

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Background: New-onset conduction abnormalities (CAs) following transcatheter aortic valve replacement (TAVR) are associated with hospital rehospitalization and long-term mortality, but available predictors are sparse. This study sought to determine clinical predictors of new-onset left bundle branch block (LBBB) and new permanent pacemaker (PPM) implantation in patients undergoing TAVR.

Methods and Results: We enrolled 290 patients who received SAPIEN 3 (Edwards Lifesciences, Irvine, CA, USA; n=217) or Evolut R (Medtronic, Minneapolis, MN, USA; n=73) from a prospective registry at Nouvel Hôpital Civil, Strasbourg, France between September 2014 and February 2018. Of 242 patients without pre-existing LBBB, 114 (47%) experienced new-onset LBBB and/or new PPM implantation. A difference between membranous septal length and implantation depth (Δ MSID) was the only predictor of CAs for both types of valves. In the multivariate analysis, PR interval and Δ MSID remained as sole predictors of CAs. The risk for adverse clinical events, including all-cause death, myocardial infarction, stroke, and heart failure hospitalization, was higher for patients with CAs as compared with patients without CAs (hazard ratio: 2.10; 95% confidence interval: 1.26 to 3.57; $P=0.004$).

Conclusions: Computed tomography assessment of membranous septal anatomy and implantation depth predicted CAs after TAVR with new-generation valves. Future studies are required to identify whether adjustment of the implantation depth can reduce the risk of CAs and adverse clinical outcomes.

Key Words: Implantation depth; Left bundle branch block; Membranous septum; Pacemakers; Transcatheter aortic valve replacement

Transcatheter aortic valve replacement (TAVR) is an established therapeutic option for intermediate and high surgical risk patients with severe aortic stenosis (AS).^{1–3} Despite increased operator experience and updated valve technologies, cardiac conduction abnormalities (CAs) remain a frequent TAVR complication.^{4–6} Clinically relevant CAs after TAVR include high-degree atrioventricular block (AVB) and other arrhythmias requiring permanent pacemaker (PPM) implantation, and new-onset left bundle branch block (LBBB).^{5,7} The incidence of these remains frequent after TAVR, seen in 31–45% of patients depending on the type of valve implanted.^{4,7,8} Recent studies have suggested that not only new PPM implantation, but also new-onset LBBB could be associated with repeat rehospi-

talization and increased mortality.^{4,9,10}

Pre-existing right bundle branch block (RBBB), first-degree AVB, self-expandable valves, intraoperative AVB, implantation depth (ID), and the length of the membranous septum (MS) have been identified as predictors of CAs.^{10–14} However, the majority of studies have been restricted to earlier-generation prostheses and the definitions of CAs have been limited to new PPM implantation. The present study was therefore designed to delineate the predictive factors of new-onset LBBB and new PPM implantation after TAVR with new-generation valves. Our main objective was to establish correlations between periprocedural imaging and clinical data and CAs.

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Methods

Study Design and Population

A total of 290 patients undergoing TAVR were enrolled from a prospective registry at Nouvel Hôpital Civil, Université de Strasbourg, Strasbourg, France, between September 2014 and February 2018. The patient flow through the study is shown in **Figure 1**. All patients had symptomatic severe AS and intermediate to high perioperative risks of death, as assessed by the logistic EuroSCORE. The indication for TAVR and approach were assessed by the local heart team. All participants gave informed written consent before the procedure and agreed to anonymous processing of their data (France 2 and France TAVI registries). The present study was performed according to the Declaration of Helsinki. Patients received aspirin (75–160 mg) and clopidogrel (300 mg loading dose and 75 mg/day maintenance dose) before TAVR, with ongoing dual antiplatelet therapy for 3 months after the procedure.

Data Source

Baseline characteristics of the enrolled patients were collected prospectively. Pre- and postprocedural ECGs were validated in all patients. RBBB and LBBB were defined by appropriate society guidelines.¹⁵ CAs were defined as new-onset LBBB and/or new PPM implantation during hospitalization.

Study Devices and Procedures

For TAVR, the balloon-expandable SAPIEN 3 prosthesis (Edwards Lifesciences, Irvine, CA, USA) or the self-expandable Evolut R (Medtronic, Minneapolis, MN, USA) was used. During the procedure, 100 IU/kg of unfractionated

heparin was administered to achieve an activated clotting time of 250–350 s. At the end of the procedure, heparin was antagonized with 100 IU/kg of protamine.

Computed Tomography Acquisition

Preprocedural ECG-gated multidetector computed tomography (CT) examinations using a 2nd or 3rd-generation 320-row CT scanners (Aquilion ONE Vision Edition, Aquilion One Genesis, Canon Medical Systems, Japan) were performed. The aortic root CT was acquired in volume mode using a retrospective ECG-gated acquisition and the following CT parameters: 16 cm width, 100 kV, gantry rotation time of 0.275 s, auto-mA maxed at 300, acquisition over 1 heartbeat.¹⁶ Acquisition was obtained after a bolus injection of 50–70 mL of iomeprol 400 mg/mL (Iomeron®, Bracco, Italy), using an automatic power injector at a rate of 3.5 mL/s, followed by a 30-mL saline chaser at a rate of 3 mL/s. The acquisition was triggered using a bolus-tracking technique with the region of interest positioned in the descending thoracic aorta and a 180 Hounsfield units threshold.

Aortic Valve Calcium Scoring

Aortic valve calcium score measurements were performed off-line on a dedicated workstation using Vitrea software in version 6.6 (Vital Imaging, USA) by the Agaston method¹⁷ and expressed in arbitrary units (AU). All scorings were performed by the same operator (K.M., cardiologist with 2 years of experience in CT).

Depth of Valves and MS Length

The depth of bioprosthesis implantation in the left ventricular outflow track was measured using a final fluoroscopic aortic angiographic acquisition after valve implantation.

Table 1. Clinical Characteristics of Participating Patients

	CA (n=114)	No CA (n=128)	P value
Age, years	84.1±7.1	83.8±7.0	0.71
Male	45 (39)	49 (38)	0.85
Body mass index, kg/m ²	27.7±6.2	26.8±5.7	0.25
Hypertension	94 (82)	112 (88)	0.27
Diabetes mellitus	35 (31)	38 (30)	0.86
Dyslipidemia	66 (58)	69 (54)	0.53
CKD	56 (49)	45 (35)	0.03
COPD	21 (18)	21 (16)	0.68
Cerebrovascular disease	12 (11)	20 (16)	0.24
Ischemic heart disease	44 (39)	64 (50)	0.07
AF history	40 (35)	36 (28)	0.24
PR interval, ms	199±44	177±30	<0.001
Presence of RBBB	19 (17)	16 (13)	0.36
Presence of LBBB	—	—	—
Duration of QRS, ms	122±44	115±34	0.21
Logistic EuroSCORE	18.9±11.7	19.0±13.3	0.98
Echocardiographic data			
Mean aortic gradient, mmHg	50±13	49±13	0.64
Aortic valve area, mm ²	0.73±0.22	0.74±0.19	0.98
LVEF, %	56±12	57±12	0.63
sPAP, mmHg	40±13	40±14	0.76
CT data			
Annulus area, mm ²	456±90	442±93	0.30
MS length, mm	7.5±2.9	8.4±2.4	0.01
Calcification in basal septum	42 (37)	44 (34)	0.69
AV calcium score, AU	3,131 (2,230–4,308)	3,004 (2,209–3,983)	0.26
MV calcium score, AU	820 (95–3,920)	751 (124–2,664)	0.50
Type of valve			0.26
SAPIEN 3	81 (71)	99 (77)	
Evolut R	33 (29)	29 (23)	
Diameter of valve (mm)			0.02
23	31 (27)	53 (41)	
26	43 (38)	50 (39)	
29	39 (34)	25 (20)	
34	1 (1)	0 (0)	
Procedure time, min	79±20	79±25	0.94
Predilatation	73 (64)	68 (53)	0.09
Postdilatation	5 (4)	11 (9)	0.19
Implantation depth (NCC), mm	6.2±2.2	5.8±2.9	0.21
Implantation depth (LCC), mm	5.3±2.7	4.5±2.4	0.02
Implantation depth (mean), mm	5.7±2.3	5.1±1.9	0.03
ΔMSID, mm	1.8±3.1	3.4±3.0	<0.001

Values are n (%), mean±SD, or median (interquartile range). AF, atrial fibrillation; AU, Agatston units; AV, aortic valve; CKD, chronic kidney disease; COPD, chronic obstructive pulmonary disease; CT, computed tomography; LBBB, left bundle branch block; LCC, left coronary cusp; LVEF, left ventricular ejection fraction; MS, membranous septum; MV, mitral valve; NCC, noncoronary cusp; sPAP, systolic pulmonary artery pressure; RBBB, right bundle branch block; ΔMSID, difference between length of MS and implantation depth.

The ID was defined as the average distance from the native aortic annulus plane (on the side of the noncoronary cusp and on the side of the left coronary cusp) to the proximal edge of the implanted valve (deepest level in the left ventricle). MS length was determined on the preprocedure CT, during systole, using the coronal view as previously described.¹³ In addition, the difference between MS length and mean ID was calculated to define the difference between them (ΔMSID).

Prosthesis Oversizing

Oversizing was calculated as follows: oversizing by area (%)=(valve area/annulus area−1)×100 for SAPIEN 3; oversizing by perimeter (%)=(valve perimeter/annulus perimeter−1)×100 for Evolut R.^{12,18} The nominal area of a fully expanded SAPIEN 3 is 409cm² for the 23-mm valve, 519mm² for the 26-mm valve, and 649mm² for the 29-mm valve. In contrast, the nominal perimeter of a fully expanded Evolut R is 72.22mm for the 23-mm valve,

Figure 2. Representative cases of CAs following TAVR. CAs, conduction abnormalities; LBBB, left bundle branch block; PPM, permanent pacemaker; TAVR, transcatheter aortic valve replacement; ΔMSID, difference between length of membranous septum and implantation depth.

81.64mm for the 26-mm valve, 91.06mm for the 29-mm valve, and 106.76mm for the 34-mm valve.

Definition of Major Adverse Cardiovascular Events (MACEs)

MACE was defined as a composite of all-cause death, myocardial infarction, stroke, and heart failure hospitalization. Outcome data were collected by serial contact with the patients and from their medical records.

Statistical Analysis

Categorical variables are expressed as numbers (%), and continuous variables are expressed as mean±SD or median and interquartile values. Differences between 2 groups were assessed with χ^2 tests for categorical variables. Unpaired Student's t-test was used to analyze continuous variables

that showed normal distributions, and the Wilcoxon test was used to analyze continuous variables with skewed distributions. Univariate and multivariate logistic regression analyses were performed to identify independent predictors of CAs. Variables with P value <0.1 in the univariate analysis were included in the multivariate analyses. Event rates over time were summarized using Kaplan-Meier estimates, and log-rank tests were used to perform comparisons between patients with and without CAs. P values of <0.05 were considered to indicate statistical significance. All analyses were performed using JMP 13 software® (SAS Institute, Cary, NC, USA).

Results

Patients' Characteristics

Of the 290 patients analyzed, 48 had pre-existing LBBB. CAs were evidenced in 114 of the remaining 242 patients (47%) (Figure 1). Baseline clinical and procedural characteristics for the patients with new-onset CAs showed higher incidence of chronic kidney disease (CKD) (49% vs. 35%, $P=0.03$) and longer PR interval (199 ± 44 ms vs. 177 ± 30 ms, $P<0.001$) (Table 1). Patients with new-onset LBBB had higher incidence of CKD (52% vs. 36%, $P=0.02$) and longer PR interval (197 ± 42 ms vs. 180 ± 35 ms, $P=0.003$). Patients requiring PPM had higher body mass index (28.5 ± 6.1 kg/m² vs. 26.7 ± 5.9 kg/m², $P=0.03$), higher incidence of chronic pulmonary obstructive disease (28% vs. 15%, $P=0.02$) and pre-existing RBBB (30% vs. 7%, $P<0.001$), longer PR interval (201 ± 43 ms vs. 186 ± 37 ms, $P=0.02$) and QRS duration (136 ± 40 ms vs. 120 ± 37 ms, $P=0.006$).

CT Measurements and Implantation Depth

Patients who presented with new CAs had a significantly shorter MS (7.5 ± 2.9 mm vs. 8.4 ± 2.4 mm, $P=0.01$) and lower implantation depth (5.7 ± 2.3 mm vs. 5.1 ± 1.9 mm, $P=0.03$) than patients without CAs (Table 1). ΔMSID was smaller in patients with CAs after TAVR (1.8 ± 3.1 mm vs. 3.4 ± 3.0 mm, $P<0.001$). There was no association between new CAs and the extent of aortic valve calcification. Although shorter MS was observed in patients with new-onset LBBB compared with patients without new-onset LBBB (7.4 ± 2.8 mm vs. 8.3 ± 2.5 mm, $P=0.02$), the length was similar between patients with and without new PPM implantation (7.9 ± 3.1 mm vs. 7.9 ± 2.7 mm, $P=0.92$). In contrast, smaller ΔMSID was significantly associated with both new-onset LBBB (1.8 ± 3.1 mm vs. 3.1 ± 3.2 mm, $P=0.002$) and new PPM implantation (1.5 ± 3.4 mm vs. 2.8 ± 3.1 mm, $P=0.007$). Representative cases are shown in Figure 2.

Predictors of Conduction Abnormalities

Figure 3 shows the inverse relationship between MS length and ΔMSID and the likelihood of CAs. Patients in the lowest quartile of MS length (≤ 6.3 mm) and ΔMSID (≤ 0.69 mm) had the highest incidence of CAs. Table 2 shows the results of univariate logistic regression analysis of predictors of subsequent CAs after TAVR. In the univariate analysis, the odds ratio (OR) for new CAs was increased in patients with CKD (OR: 1.78; 95% confidence interval [CI]: 1.06 to 2.98; $P=0.03$), longer PR interval (OR: 1.02; 95% CI: 1.01 to 1.03; $P<0.001$), shorter MS (OR: 0.88; 95% CI: 0.80 to 0.97; $P=0.01$), greater ID (OR: 1.15; 95% CI: 1.01 to 1.31; $P=0.02$), and decreased ΔMSID (OR: 0.84; 95% CI: 0.76 to 0.92; $P<0.001$).

Multivariate logistic regression analysis for the preprocedural prediction model revealed PR interval (OR: 1.01; 95% CI: 1.00 to 1.03; $P=0.006$) and MS length (OR: 0.84; 95% CI: 0.73 to 0.97; $P=0.02$) as independent predictors of CAs after TAVR (Table 3). In the postprocedural prediction model, PR interval (OR: 1.02; 95% CI: 1.00 to 1.03; $P=0.006$) and ΔMSID (OR: 0.77; 95% CI: 0.67 to 0.89; $P<0.001$) were independent predictors of CAs.

Difference Between Types of Valve

Baseline and procedural characteristics of the subgroups of patients with SAPIEN 3 and Evolut R valves are shown in the Supplementary Table 1 and Supplementary Table 2. Among all patients, 180 (74%) received a SAPIEN 3 prosthesis and 62 patients (26%) received a Evolut R prosthesis. In each subgroup, significantly smaller ΔMSID was apparent in patients with CAs (2.0 ± 3.2 mm vs. 3.5 ± 2.8 mm, $P=0.002$ and 1.1 ± 2.9 mm vs. 3.2 ± 3.7 mm, $P=0.02$, respectively), while MS length did not reach significant difference between patients with and without CAs (7.5 ± 3.0 mm vs. 8.3 ± 2.4 mm, $P=0.06$ and 7.5 ± 2.6 mm vs. 8.7 ± 2.5 mm, $P=0.08$, respectively). Similarly, prosthesis oversizing was not associated with CAs in the 2 subgroups.

Table 2. Univariate Analysis for Prediction of New Conduction Abnormalities

	OR (95% CI)	P value
Age, years	1.01 (0.97–1.04)	0.71
Male	1.05 (0.63–1.76)	0.85
Body mass index, kg/m ²	1.03 (0.98–1.07)	0.25
Hypertension	0.67 (0.33–1.37)	0.27
Diabetes mellitus	1.05 (0.61–1.82)	0.86
Dyslipidemia	1.18 (0.71–1.96)	0.53
CKD	1.78 (1.06–2.98)	0.03
COPD	1.15 (0.59–2.24)	0.68
Cerebrovascular disease	0.64 (0.30–1.37)	0.25
Ischemic heart disease	0.63 (0.38–1.05)	0.08
AF history	1.38 (0.80–2.38)	0.24
PR interval, ms	1.02 (1.01–1.03)	<0.001
Presence of RBBB	1.40 (0.68–2.87)	0.36
Presence of LBBB	–	–
Duration of QRS, ms	1.00 (1.00–1.01)	0.21
Logistic EuroSCORE	1.00 (0.98–1.02)	0.98
Echocardiographic data		
Mean aortic gradient, mmHg	1.00 (0.99–1.02)	0.64
Aortic valve area, cm ²	0.98 (0.28–3.44)	0.98
LVEF, %	0.59 (0.07–5.07)	0.63
sPAP, mmHg	1.00 (0.98–1.03)	0.76
CT data		
Annulus area, mm ²	1.00 (1.00–1.00)	0.30
MS length, mm	0.88 (0.80–0.97)	0.01
Calcification in basal septum	1.11 (0.66–1.89)	0.69
AV calcium score, per 100	1.02 (1.00–1.04)	0.09
MV calcium score, per 100	1.01 (1.00–1.02)	0.13
Type of valve		
SAPIEN 3	0.72 (0.40–1.28)	0.26
Evolut R	1.39 (0.78–2.48)	0.26
Procedure time, min	1.00 (0.99–1.01)	0.94
Predilatation	1.57 (0.94–2.63)	0.09
Postdilatation	0.49 (0.16–1.45)	0.20
Implantation depth (mean), mm	1.15 (1.01–1.31)	0.02
ΔMSID, mm	0.84 (0.76–0.92)	<0.001

CI, confidence interval; OR, odds ratio. Other abbreviations as in Table 1.

Prognostic Effect of Conduction Abnormalities

At 3 years after TAVR, the incidence of MACE was 53.4% in patients with new CAs and 30.1% in patients without new CAs (Figure 4). The hazard ratio (HR) of MACE during the 3 years after TAVR was 2.10 (95% CI: 1.26 to 3.57; $P=0.004$). In a subanalysis, new-onset LBBB and new PPM implantation were both related to increased MACE rates at 3 years (HR: 1.88; 95% CI: 1.13 to 3.13; $P=0.01$ and HR 1.90; 95% CI: 1.07 to 3.25; $P=0.03$, respectively) (Supplementary Figure 1). The Kaplan-Meier curves for each event included in the combined endpoint are shown in Supplementary Figures 2–4. CAs were associated with all-cause death (HR 2.07; 95% CI: 1.07 to 4.13; $P=0.03$), stroke (HR 3.08; 95% CI: 1.16 to 9.62; $P=0.02$), and heart failure hospitalization (HR 2.17; 95% CI: 1.11 to 4.41; $P=0.02$) during the 3-year follow-up.

Table 3. Multivariate Analyses for Prediction of New Conduction Abnormalities

	OR (95% CI)	P value
Preprocedure		
CKD	1.83 (0.89–3.75)	0.10
Ischemic heart disease	0.79 (0.39–1.60)	0.51
PR interval, ms	1.01 (1.00–1.03)	0.006
MS length, mm	0.84 (0.73–0.97)	0.02
AV calcium score, per 100	1.01 (0.99–1.04)	0.38
Pre- and postprocedure		
CKD	1.74 (0.82–3.71)	0.15
Ischemic heart disease	0.84 (0.40–1.77)	0.65
PR interval, ms	1.02 (1.00–1.03)	0.006
AV calcium score, per 100	1.01 (0.98–1.04)	0.46
ΔMSID, mm	0.77 (0.67–0.89)	<0.001
Predilatation	1.98 (0.92–4.30)	0.08

Abbreviations as in Table 1.

Discussion

The aim of the present study was to investigate predictors of new-onset LBBB and/or new PPM implantation specifically in patients undergoing TAVR with new-generation valves. The major findings were: (1) the rate of CAs following TAVR was 47% overall; (2) similar incidence of CAs was observed in patients with SAPIEN 3 or Evolut R valves; (3) ΔMSID was the strongest predictor of CAs with new-generation valves; and (4) compared with patients with no CAs, the risk of adverse clinical events was increased in patients with new CAs.

Possible Mechanisms of Conduction Abnormalities

Previous studies have indicated that direct injury of the intraventricular conduction system during valve implantation is the possible mechanism of CAs.^{19,20} The atrioventricular (AV) conduction axis originates from the compact AV node and is located along the inferior edge of the MS.^{21,22} The axis gives rise to the left bundle branch along the crest of the muscular ventricular septum. Therefore, a direct injury during valve implantation is likely to provoke new-onset LBBB or high-degree AVB following TAVR.

Incidence of Conduction Abnormalities

The occurrence of new-onset CAs after TAVR has had varying incidence reported across studies.^{7–13} The incidence of new-onset LBBB has been more frequent when the self-expandable CoreValve system (Medtronic) is used (18–65%), compared with 4–30% reported with the use of the balloon-expandable Edwards SAPIEN/SAPIEN XT valve (Edwards Lifesciences).⁵ Consistently, the rate of PPM implantation has also been higher with the CoreValve system than with SAPIEN/SAPIEN XT valve (25–28% vs. 5–7%). For the newer generation valves, several studies have indicated that the occurrence of new-onset CAs was higher with SAPIEN 3 implantation than with the SAPIEN XT. De Torres-Alba et al reported that both the incidence of new PPM implantation (19.1% vs. 12.2%, $P=0.046$) and new-onset LBBB (13.0% vs. 10.8%, $P=0.492$) were higher for SAPIEN 3.⁸ Similarly, the SOURCE 3 Registry (SAPIEN Aortic Bioprosthesis European Outcome) also demonstrated a higher rate of new PPM

Figure 4. Cumulative incidence of adverse clinical outcomes up to 3 years after transcatheter aortic valve replacement for patients with and without CA. CA, conduction abnormality; CI, confidence interval; MACE, major adverse cardiovascular events.

implantation for SAPIEN 3 (12% vs. 6%).²³ In contrast, CAs after TAVR with Evolut R are reported to be less frequent than with CoreValve (new-onset LBBB: 11.7% vs. 44.7%, $P<0.05$; new PPM implantation 10.9% vs. 21.6%, $P=0.01$).²⁴ It is noteworthy that our data showed similar rates of CAs between SAPIEN 3 and Evolut R. However, the results should be interpreted with caution because our study was not powered to compare these 2 types of valves. Non-significant increase in the rate of CAs was observed in patients with Evolut R as compared with patients with SAPIEN 3 (53% vs. 45%, $P=0.26$), suggesting that larger studies are warranted to confirm our findings.

Although the timing of PPM implantation and indications vary among institutions, the CAs rate in the present study (47%) was similar to that in a recent study reported by Jørgensen et al, which suggested that new-onset LBBB and new PPM implantation occurred in 45% of patients undergoing TAVR with various types of valves.⁴

Predictors of Conduction Abnormalities

Identified risk factors for new-onset LBBB include self-expandable valve, ID, and overexpansion of the native aortic annulus.^{20,25,26} Similarly, the predictors of PPM implantation include the same factors but also baseline RBBB and first-degree AVB.^{11,27–30} Furthermore, Hamdan et al demonstrated that shorter MS and Δ MSID were related to new PPM implantation following TAVR in a registry including 73 self-expandable valve recipients (6.5 ± 2.6 mm vs. 9.3 ± 2.9 mm, $P<0.001$ and -1.2 ± 4.2 mm vs. 3.7 ± 4.3 mm, $P<0.001$, respectively).¹³ Likewise, Maeno et al studied a cohort of 240 SAPIEN 3 recipients and suggested a relationship between MS length or Δ MSID and the need for PPM implantation (6.4 ± 1.7 mm vs. 7.7 ± 1.9 mm, $P<0.001$ and 0.6 ± 2.9 mm vs. -2.5 ± 2.4 mm, $P<0.001$, respectively).¹² In contrast, Oestreich et al failed to identify an association of MS length or Δ MSID with new-onset LBBB or new PPM implantation (7.9 ± 2.0 mm vs. 7.2 ± 2.0 mm, $P=0.20$ and 0.5 ± 4.0 mm vs. 1.1 ± 4.0 mm, $P=0.48$, respectively) in a series

of 102 SAPIEN 3 recipients.³¹ These differences across studies may be partly explained by variations in the location of the MS itself relative to the plane of the visual basal ring.²² The MS is also known to vary in size,^{32,33} a variation recognizable using CT analysis,³⁴ and there is variation in the rotation of the aortic root due to the underlying ventricular mass.³⁵ Accordingly, discrepancies may be found among studies especially when the sample size is small. The present study included 242 TAVR patients with preprocedural MDCT data, which is the largest study to assess the periprocedural predictors of CAs, including MS length. Our study underlined that the clinically relevant measurement is not the height of the MS, but rather the relationship of the depth of the implanted valve relative to its inferior aspect. Interestingly, our study demonstrated that Δ MSID was related to CAs with both new-generation balloon- and self-expandable valves.

Although prior studies have demonstrated that calcification of the left coronary cusp or the device-landing zone is predictive of PPM implantation after TAVR,^{36,37} we could not find a relationship between the aortic valve calcium score and CAs. In the present study, the spatial distribution of the calcification was not considered, which could be an explanation for the observed differences. Fujita et al revealed that the total calcium was not related to new PPM implantation after TAVR, but there was a strong correlation between an elevated level of left coronary cusp calcification and PPM implantation.³⁷ Shifting the valvuloplasty balloon and the TAVR valve away from the left coronary cusp towards the right/noncoronary cusp region was considered a potential mechanism of injuring the conduction pathway.

Clinical Outcomes of New-Onset LBBB and New PPM Implantation After TAVR

Prior studies with more than 1 year of follow-up have failed to show an association between new PPM implantation after TAVR and all-cause death. However, those studies included patients with new-onset LBBB in the no PPM

implantation group. Given that LBBB increases the risk of MACE,^{38,39} the inclusion of patients with LBBB in a comparator group might be why previous studies did not identify an association between new PPM implantation following TAVR and long-term adverse outcomes. Recently, Jørgensen et al reported that new bundle branch block and new PPM implantation were both associated with late all-cause death after TAVR.⁴ Our findings were consistent with these observations, as patients with new-onset LBBB or new PPM implantation in our cohort had developed cardiovascular events 2-fold at the 3-year follow-up.

Study Limitations

First, the decision to implant a PPM was ultimately at the discretion of the attending physician. The decision for choosing to implant a PPM may differ among physicians and even more widely among institutions. To account for the effect of such variations on the outcome parameter, we selected not just the PPM rate as the dependent variable but also LBBB, for which there is no physician- or institution-dependent variation. Second, a modest proportion of patients (n=171) were excluded from initial enrollment, mostly due to the absence of analyzable CT images. Third, the presence of CAs was evaluated during the patient's admission, meaning that some patients might have been misclassified because of only transient LBBB.

Conclusions

AMSID was the most powerful predictor of CAs following TAVR with new-generation valves. New-onset CAs were associated with adverse clinical outcomes at 3 years. The procedural aspect of higher valve implantation, relative to MS length, may mitigate the occurrence of CAs following TAVR.

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Disclosures

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IRB Information

The approval for this study was obtained from the France 2 study: 911262.

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Supplementary Files

Please find supplementary file(s);
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